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Experiments in Microwave Exposure in the Rat Body Temperature, Serum Chemistry, and the Use of Chemical Restraint

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16. ABSTRACT

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Various injectable anesthetics were evaluated as restraints in microwave exposure experiments. Colonic temperature was a measure of the response to exposure. Immobilization was accompanied by decreased colonic temperature. After exposure to 30 mW/cm^2 for 90 min, mean colonic temperatures in chemically restrained rats ranged from 39-40.4°C, and increased up to 5.5°C from anesthetically-depressed pre-exposure levels. Limitations on the use of chemical restraint for this kind of experiment and on the use of multi-value serum chemistry screening tests were determined in the course of this research.

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EXPERIMENTS IN MICROWAVE EXPOSURE IN THE RAT:
Body Temperature, Serum Chemistry, and the Use of Chemical Restraint

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FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis and the toxicology of pesticides as well as other chemical pollutants. The Laboratory participates in the preparation of air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is primarily responsible for providing the health basis for non-ionizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of

expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care for persons at risk in their health.

Applications for microwave equipment are being found in many different technologies. Ovens, heaters, telephones, television, and satellite communications are areas where this equipment has become ubiquitous.

Understanding the consequences of microwave exposure in humans depends largely on the development of a base of knowledge derived from animal experimentation. This report is an initial step in building a data base to use in evaluating the effects of microwave radiation in human health.

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ABSTRACT

A series of experiments was conducted in rats to observe their responses to 2450-MHz (CW) microwaves. Colonic temperatures were measured after exposure to 0, 20, or 30 mW/cm^2 for up to 4 h. Exposures of 30 mW/cm^2 caused a regular and significant increase in colonic temperature. A plateau of 38.9° was reached in 90 min and maintained for the rest of the 4-h exposure. Rats exposed to 20 mW/cm^2 for 4 h exhibited patterns of colonic temperature similar to sham-exposed rats. Acclimation for 90 min had no effect on these patterns. Measurements (or calculations) of 26 serum chemistry values produced similar results in rats exposed to 30 mW/cm^2 for 120 min and rats exposed to 0 or 20 mW/cm^2 for 120 min. Only serum corticosterone was significantly increased in the dose-related response of naive rats to this acute exposure.

Various injectable anesthetics were evaluated as restraints in microwave exposure experiments. Colonic temperature was a measure of the response to exposure. Immobilization was accompanied by decreased colonic temperature. After exposure to 30 mW/cm^2 for 90 min, mean colonic temperatures in chemically-restrained rats ranged from 39-40.4°C, and increased up to 5.5°C from anesthetically-depressed pre-exposure levels. Limitations on the use of chemical restraint for this kind of experiment and on the use of multi-value serum chemistry screening tests were determined in the course of this research.

SECTION 1

INTRODUCTION

Extrapolating from the results of microwave-exposure studies to human health effects depends mainly upon the development of a base of knowledge from laboratory animal experimentation. Because such experiments are conducted under a great variety of conditions, attempts at replication in other laboratories often fail. Variation in microwave-exposure conditions may account for some of these failures, but experimental technique may also vary among laboratories in ways less obvious to the researcher. Apparently, even minor variations in the techniques of handling and restraint can cause untoward reactions and results in laboratory animals.

The physiologic consequences of exposure to microwave irradiation have been measured by many methods. Perhaps the most common standard of assessment of an effective level of exposure is the measurement of body or colonic temperature (Phillips et al., 1975; Ely and Goodman, 1956; Tell, 1978; Cleary, 1974; Pazderova-Vejlupkova and Josifko, 1979; Berman et al., 1978; Berman et al., 1981; Lu et al., 1977; Polson et al., 1974). Temperature is usually taken just before and just after an exposure. Measurements taken during an exposure have had limited interpretability because of errors introduced by metallic temperature sensors. However, reliance on post-exposure body temperature

values is not justified without measurements of temperature variations encountered during the exposure itself.

The rat was chosen as the experimental subject for these studies because it is one of the most commonly employed whole animal models in the microwave bioeffects field as well as in this laboratory. For instance, in the review of O'Connor (1980) of the results from 17 reports of microwave teratogenesis in animals, about half of the studies were conducted in rats. Another important rationale for the use of rats in these studies is that the rat may randomly orient itself in a 2450-MHz frequency microwave field without the whole-body specific absorption rate (SAR) being significantly affected; whereas, in mice, the SAR varies according to orientation (Gage et al., 1977).

This report presents data from rats exposed to microwave radiation under "typical" experimental conditions. These results are intended for use as reference or baseline data; they are not to be interpreted as biological effects of microwave exposure.

SECTION 2

CONCLUSIONS

When naive young adult male rats are exposed to 2450-MHz microwaves at a power density of 30 mW/cm^2 with a duration of up to 4 h, their colonic temperatures rise to 39°C. Twenty mW/cm^2 causes no obvious temperature increase in rats. However, acclimation to the same conditions, without microwaves, does not alter the pattern of increased temperature. Except for a significant rise in corticosterone concentration, serum chemistry values in animals exposed to 30 mW/cm^2 are not acutely altered.

Anesthetics are not recommended for restraint in animals during microwave exposure unless it is appreciated how they limit normal thermal physiologic mechanisms.

SECTION 3

RESULTS AND DISCUSSION

Body Temperature

Experiments 1 and 2 (Table 1) measured body temperatures in the rat after exposures to varying power densities and durations up to 4 h. Experiment 1 (Tables 1 and 2) compared power densities of 0, 20, and 30 mW/cm^2, the latter being a common high level of power density used in rats in this laboratory. The curve (Figure 1) of mean body temperatures after increasing exposure durations showed a rapid (15 min) response to this high power density. Within 60 min, the mean temperature had risen to essentially 39°C, and remained there during the longer exposures for up to 4 h. Extended exposure to a power density of 30 mW/cm^2 produced a response within the thermal physiologic capacity of the rat, as evidenced by a plateau of body temperature. How long the plateau could be maintained in the face of continued absorption of this energy (SAR $\cong$ 6.6 mW/g; Gage et al., 1977) is not known and is beyond the scope of this report.

On the other hand, a power density of 20 mW/cm^2 applied under identical conditions had virtually no effect on body temperature. The curve (Figure 1) of body temperature at various exposure durations at this power density

(SAR ≅ 4.4 mW/g) approximates that of sham exposures, except for the initial period of up to 1 h, in which lower body temperatures were registered for shams. Apparently, 20 mW/cm^2 did not constitute a significant absorption of energy by these rats, and was conveniently handled by these animals.

The rats in all these experiments were naive to the procedures used. There are limitations on the interpretation of responses in rodents in naive situations. To help avoid these obstacles, some researchers use a period of acclimation to the experimental techniques on the basis that a stress response to a new experimental situation can generate a considerable amount of physiologic "noise". In turn, it is possible to misinterpret the "noise" as being the rats' response to microwave exposure (Michaelson et al., 1975) and, thus, a more subtle response may be entirely masked. To some degree, acclimation was provided in Experiment 2 (Table 3, Figure 2). Naive rats were acclimated to the exposure conditions (handled, caged in the constrainers, and exposed in the environmental chambers at 0 mW/cm^2) for 90 min. Body temperatures were not measured, but were expected to follow a decreasing curve like that of rats sham-irradiated in Experiment 1 (Figure 1). What seems like dramatic decrease in the sham-irradiated rats' temperatures results from the contrast with their initially high mean temperatures (approximately 38.2°C; pre-exposure values, Figure 1). Those high temperatures were caused by the excitement from being awakened in the maintenance cage, and handled and transported to the exposure facility. Within 60 min, the mean temperature of sham-irradiated rats fell below 37°C, and the animals were quiet and usually dozing.

Rats that were acclimated for 90 min, then microwave-irradiated for 90 min at 30 mW/cm^2 (Experiment 2, Figure 2, Table 3) had essentially the same body temperatures as those irradiated with 30 mW/cm^2 with no acclimation (Figure 1, Table 2). Acclimation of 90 min did not appear to influence the final body temperature reached by rats exposed to 30 mW/cm^2. Even when acclimation reduced the temperature to <37°C and at least 1.0°C below the initial pre-exposure value, the final temperature still reached levels seen in non-acclimated rats. Therefore, the temperature at the beginning of the exposure did not appear to be a significant influence on the final temperature after exposure to microwave radiation.

Anesthetics

The measurements made in Experiments 1 and 2 (Table 1) were taken in animals that had normal thermoregulation. The rats used in Experiment 3 were administered six anesthetics (Figures 3-8), all of which altered physiologic thermal control. Used commonly in veterinary and laboratory animal procedures, these chemical restraints were explored for their suitability for microwave experiments (immobilization for 1-2 h, without effect on physiologic thermal mechanisms). The compounds used were anileridine, a synthetic narcotic; chloral hydrate and pentobarbital, both CNS anesthetics; promazine, a tranquilizer; and ketamine and xylazine, both dissociative drugs.

The use of chemical restraint in animals undergoing microwave exposure was suggested by Majde (1978). Though Majde was specifically discussing the use of dissociative drugs in monkeys, the broader implication was that shivering and

sweating activities are not abated and thermoregulation remains normal in animals under the influence of ketamine or xylazine.

We administered the chemicals at doses suggested in the literature on laboratory animals or veterinary medicine (Hughes, 1981; Barnes and Eltherington, 1966; Merck, 1973; Jones et al., 1977; Soma, 1971; Green, 1979). These doses provided starting points from which the lowest dose producing practical immobilization was determined. For example, the recommended anesthetic dose of pentobarbital in the rat is 25-40 mg/Kg i.p. (Hughes, 1981) or 50 mg/Kg i.p. (Barnes and Eltherington, 1966). We found that 35 mg/Kg i.p. was an effective anesthetic dose. The effective tranquilizing dose for promazine in the rat is given as 120 mg/Kg i.p. (Barnes and Eltherington, 1966) or 1-20 mg/Kg i.m. (Hughes, 1981). The deepest immobilization was obtained from 40 mg/Kg i.m. Recommended doses of anileridine in animals were not available, as the drug was not recommended for veterinary use. (This drug is no longer manufactured commercially.) The doses of drugs used to obtain the greatest immobilization and least response to touch (Figures 3-8) were: anileridine, 10 mg/Kg s.c.; chloral hydrate, 300 mg/Kg i.p.; ketamine, 160 mg/Kg i.m.; pentobarbital, 50 mg/Kg i.p.; promazine, 40 mg/Kg i.m.; and xylazine, 0.75 mg/Kg i.p.

Anileridine caused little loss of wakefulness and produced little immobilization (Figure 3). The response to touch was still present during the measurement of temperature, but quiet handling was possible. Colonic temperatures during the test period remained as high as in active animals, and we suspect that some mild hyperthermia had resulted from the dose.

Chloral hydrate produced profound sleep and immobilization at 300 mg/Kg (Figure 4) and depressed the response to touch for more than 90 min after injection. Colonic temperature means were steadily depressed by this drug, falling below 36°C near the end of the effective period, a decrease of 1.5°C (Figure 4). All anesthetized rats were observed while in open-topped plastic cages shaped like shoe boxes with pine-shaving bedding; without this insulation, temperatures might have decreased even further than observed.

Ketamine depressed effectively the response to touch, but caused almost no immobilization (Figure 5). The body temperature depression was, like chloral hydrate, approximately 1.5°C. The depression of temperature with no effective immobilization suggests that ketamine alone is not a useful drug for restraint during microwave exposure.

Pentobarbital is very commonly used as an animal anesthetic and has been thoroughly researched. Rats responded with deep and constant immobilization and with a lack of response to touch for at least 90 min after a single i.p. injection of 35 mg/Kg (Figure 6). During this period, body temperatures were down to 35.5°C, a loss of 2-2.5°C. All three measures (response, sleep, temperature) were affected for almost 3 h. The use of male rats and the bedding may have offset each other in this experiment: male rats are reported to sleep longer than females, and pine-shaving bedding is reported to reduce the drug-induced sleep time (Hughes, 1981).

Promazine (chlorpromazine; Sparine®) was relatively ineffective in reducing touch-response (Figure 7). There was a long delay (> 60 min) before any depth

of sleep occurred, and the sleep was erratic. The drug was effective (within its small effective dose range) for the entire 3-h period. Mean body temperature by that time were almost 2°C below initial temperature. Promazine's delayed and erratic effect suggests that this drug is not useful as a restraint for microwave exposures.

Xylazine was ineffective. No sleep (immobilization) occurred, and response to touch was normal (Figure 8). Even so, temperatures at 90 min after injection were 2°C below initial temperatures.

When rats were given one of the four chosen chemicals, and then irradiated with 2450-MHz microwaves at a power density of 30 mW/cm^2 for 90 min, rectal temperatures showed a sharp increase (Table 4). Mean temperatures at the end of exposure were 39 to > 40°C. These values were as much as 5.5°C above those encountered with the drug but without microwave exposure (Table 5). Because body temperatures also exceeded those of rats in similar microwave-exposure situations (Experiment 1; Table 2), we suspect that all the tested drugs interfered with normal thermal regulation. Therefore, we do not recommend the use of these drugs as restraining agents in microwave exposure when body temperatures are used to determine physiologic response, unless the compromised thermal regulatory functions are taken into account.

Serum Chemistries

Chemical analyses of serum constituents also are common experimental techniques for the assessment of physiologic response to microwave exposure

in laboratory animals (Cleary, 1974; D'Andrea and Gandhi, 1981; Guillet and Michaelson, 1977; Lovely et al., 1977; Wangemann and Cleary, 1976; Dumas et al., 1980). The use of serum chemistries in this respect may well increase because of the economy and convenience associated with modern automated analytic techniques. For example, the SMAC-24 tests used in Experiment 4 reported 24 replicated values and ratios of 21 constituents on one serum sample of less than 1-ml volume. Only one additional milliliter was required to do the serum T_3 and T_4 and corticosterone tests using radioimmune assay techniques. The cost of this battery of tests is insignificant when compared to the cost incurred by manual analyses used just a few years ago.

Because these tests are convenient and relatively economical, one can lose sight of their limited applicability. Data values are often subjected to no more evaluation than statistical tests of confidence (t-test; analysis of variance). A warning of the hidden problems associated with indiscriminate use of serum chemistry values was presented by Mitruka and Rawnsky (1977). They recommended the consideration of age, sex, strain, environment, and physiologic status (diurnal cycle, postural stance, age) of the animal when interpreting values derived from blood. They also stressed that in human medicine, in which these batteries of tests find their most effective use and for which they were designed, the test results should be confined to decisions of diagnosis, treatment, and prognosis, and that the critical aspect is interpretation. Also, any interpretation should be strongly limited when there are no baseline values derived from the same population that can also be extrapolated to other populations.

More important, perhaps, is the suggestion by Mitruka and Rawnsky (1977) that a Bayesian approach should be used in interpreting these diagnostic tests. Paraphrased for toxicologic research, e.g., microwave-exposure studies, the requirements might be: (a) clear cut definition of the disease (effect) being sought; (b) delimitation of the stage of the effect being evaluated by the test; and (c) knowledge of true-positive and true-negative results of the test. More simply stated, one should have a firm prior hypothesis!

Statistically, the determination of 21, 24, or more values at once in one subject is expected to very much reduce the independence of these values. Appropriate adjustments should be made when applying ordinary statistical tests. In our research, the method we used extended acceptable differences to $p \leq .01$ (Table 8). Another related aspect of statistical methodology is that serum chemistry values may not assume normal distributions, and statistical tests which require normality (t-test) may not be applicable here. Bilirubin values, for instance, were expressed only as 0, 0.5, 1.0, or 1.5; chi-square statistics are more appropriate than t-test statistics in the cases of creatinine and bilirubin values (Tables 7-10).

Only three serum constituents (alkaline phosphatase, corticosterone, and creatinine, Table 6) exceeded the limitations of the analytic criteria ($p \leq .01$). However, further evaluative tests should be put to these values. It is reasonable to expect the values to follow some consistent direction or trend. The application of microwaves should cause an increasing or decreasing trend in the results. Biphasic (up-then-down, or down-then-up) trends are not to be reasonably expected from increasing exposure. However, alkaline phosphatase increased

from 250 mg/dl serum in the 0 mW/cm^2 group to 315 in the 20 mW/cm^2 group, and then decreased in the 30 mW/cm^2 group (Table 6). Such an up-then-down pattern is unexpected and more than difficult to rationalize as resulting only from exposure to microwaves. Serum alkaline phosphatase increases in response to extrahepatic blockage or to bone disease (Wells and Halsted, 1967). As these values did not continue to increase with the increased power density, the rationale of microwave exposure as the cause of the observed changes in serum alkaline phosphatase does not bear scrutiny.

Serum creatinine values, on the other hand, did show a trend in one direction (Table 7). They showed a sharp increase in animals exposed at 20 mW/cm^2 and 30 mW/cm^2 as compared to sham animals ($p = .0001$). Because no physiologic rationale is available to explain the increase in serum creatinine, we suspect that we observed a sampling error, especially in the rather low values of 0.3-0.4 mg/dl in the sham group. The values are well within normal limits for rats (Mitruka and Rawnsky, 1977); when the values of all animals are averaged (0.47 mg/dl), the average is very close to the mean of normal rats (0.45 mg/dl; Mitruka and Rawnsky, 1977).

A corticosterone increase due to microwave irradiation (Table 6) is the only one of the statistically different serum constituents which may lend itself to a reasonable physiologic rationale. Adrenal corticoid secretion is stimulated as a result of handling of animals in naive situations (Riley and Spanckman, 1978). As the exposure to 30 mW/cm^2 was sufficient to increase colonic temperatures to almost 39°C (Table 2), it may also have caused an

increase in adrenal corticosteroid hormone secretion. The additional response to 30 mW/cm^2 exposure, as a stress reaction, may account for the more than doubling of corticosterone concentration in the high dose group as compared to shams (260 vs 116 µg/ml, respectively, Table 6). This type of adrenal reaction was similarly interpreted in the report of Wangemann and Cleary (1976) as a result of significant microwave exposure.

SECTION 4

EXPERIMENTAL PROCEDURES

The animals in these studies were naive CD male rats, 90-110 days of age. In each study, the rats were exposed to either sham conditions or microwave radiation in an environmental chamber in a 2450-MHz (CW) anechoic facility (Blackman et al., 1975; Elder and Ali, 1975). During treatment each rat was contained in a slatted acrylic cage, 18 cm long x 10 cm high (Berman et al., 1981). The long axis of the cage was oriented parallel to the H-field vector, over a polystyrene foam tray (Western Foam Pac, Inc., Greensboro, NC) to catch body wastes. Ambient air conditions were 22.2 ± 0.2°C, 50 ± 5% relative humidity, and 0.1 m/s airspeed. Sham-irradiated subjects were treated identically, but without radiation, in an environmental chamber adjacent to the anechoic chamber. Airflow was common to both environmental chambers.

Specific experimental conditions, as they varied among the experiments of this study, are outlined in Table 1. In Experiment 1, naive rats were microwave-irradiated once with varying power densities and durations. During irradiation, two cages with rats were positioned in the environmental chamber so that their centers were 22 cm from the center of the field and 44 cm from each other.

Immediately before and after exposure, the colonic temperature of each rat was measured using an electronic thermometer (Model 46 TUC and Model 402 probe; Yellow Springs Instruments Co., Inc., Yellow Springs, OH). Values taken just after exposure are expressed as means and error terms in Table 2. Curves of these values are in Figure 1.

Identical experimental methods were used in Experiment 2, except that each naive rat was acclimated for 90 min in its exposure environment before irradiation for 90 min. In this experiment, colonic temperatures were measured before the acclimation period and after the irradiation period. The numerical results of this experiment are in Table 3. Graphic presentation is in Figure 2, where these results can be compared with relevant data (0 and 30 mW/cm^2) of Experiment 1. Statistical analyses (*t*-test) were made only between the pre-exposure temperatures of Experiment 2 and post-exposure temperatures of rats that had undergone a 90-min exposure of 30 mW/cm^2 in Experiment 1.

In Experiment 3, animals were injected with one of a variety of general anesthetics, tranquilizers, or disassociative drugs (doses are mg/kg body weight):

1) anileridine* (Merck, Sharpe, and Dohme, West Point, PA), 5, 10, and 20 s.c.
2) chloral hydrate, 200 and 300 i.p.
3) ketamine (Parke-Davis and Co., Detroit, MI), 80 and 160 i.m.
4) pentobarbital (Nembutal, Abbott Labs, N. Chicago, IL), 20, 35, 50, and 65 i.p.

*No longer commercially available.

5) promazine (Sparine, Wyeth Laboratories, Philadelphia, PA), 10, 20, and 40 i.p.

6) xylazine (Rompun, Haver-Lockhart Laboratories, Shawnee, KA), 0.5, 0.75, 1, and 1.25 i.p.

At 5-, 10-, and 15-min intervals after injection, and at 15-min intervals thereafter up to 3 h, observations were made of the rat's depth of sleep, responsiveness to being touched, and colonic temperature. For defining the depth of sleep induced by the drug, the rat was considered asleep if it appeared immobile, and awake otherwise. Responsiveness was observed when the animal was touched as its temperature was taken and was graded as follows: 0 = excited, 1 = subdued, 2 = immobilized, 3 = not responsive. Values for each group of rats are expressed for colonic temperature, percent asleep, and degree of responsiveness. The results are shown in Figures 3-8.

Appropriate doses of four of the six drugs, selected on the basis of their greater usefulness in immobilizing rats for 180 min, were administered before an exposure to microwaves at a power density of 30 mW/cm^2 for 90 min. Animals were irradiated identically to those in Experiment 1, and colonic temperatures were measured just before and just after exposure. (See Table 4.) Their relative values are shown in Table 5.

Experiment 4 (Table 6) was a study of serum chemistry values immediately after a 120-min exposure to 0, 20, or 30 mW/cm^2. Rats were exposed to microwave radiation in pairs of cages as in Experiment 1. Within 5 min after treatment the animal was killed with CO_2 gas, the blood was drawn directly from the vena

cava, and the serum was separated. Chemical analyses of serum were performed using SMAC-24 procedures (Smith Kline Clinical Laboratories, Inc., Creve Coeur, MO). SMAC-24 includes the following direct analyses: blood urea nitrogen (BUN), creatinine, sodium, potassium, chloride, uric acid, calcium, phosphorus, cholesterol, triglyceride, direct bilirubin, indirect bilirubin, alkaline phosphatase, creatinine phosphokinase, lactate dehydrogenase, glutamic-oxaloacetic transaminase, glutamic-pyruvic transaminase, albumin, globulin, and total protein. Indirect calculations were made to determine BUN/creatinine ratios, total bilirubin, and albumin/globulin ratios. Glucose was also analyzed, but is not included in this report. Serum T_3, T_4, and corticosterone were determined using radioimmunoassay techniques.

Rats used in these blood collections (Experiment 4) were not deprived of either food or water except during their exposure period of 2 h. The results of these analyses are in Table 6, where the statistical parameters of each serum variable are listed by power density for those animals bled immediately after exposure. Using analysis of variance techniques (ANOVA), the results for each serum variable were compared for effects of exposure to different power densities. Because there were so many variables that could be statistically analyzed, the acceptance of statistical differences was limited to only those analyses which yielded $p \leq 0.01$. Because of the non-continuous nature of their values, serum creatinine and bilirubin are listed separately in Tables 7-10.

TABLE 1. SUMMARY OF EXPERIMENTS 1-4
ON THE EFFECTS OF MICROWAVE EXPOSURE IN RATS

Experiment 1: The effects of power density and length of exposure on colonic temperature.

Power densities: 0, 20, 30 mW/cm^2.
Length of exposure: 15, 30, 60, 120, 180, or 240 min.
Exposure array: two cages.
Biologic assay: colonic temperature immediately before and after exposure.

Experiment 2: The effect of pre-exposure acclimation on the response of colonic temperature to irradiation.

Power densities: 0 for 90-min acclimation followed by 30 mW/cm^2 for 90 min exposure.
Exposure array: two cages.
Biologic assay: colonic temperature immediately before acclimation and after exposure.

Experiment 3: Chemical restraint of rats during exposure to microwaves.

Power densities: 0 and 30 mW/cm^2.
Length of exposure: 90 min.
Exposure array: one cage.
Biologic assay: colonic temperature just after exposure; anesthetic or sedative characteristics of six chemicals.

Experiment 4: The effect of microwaves on the values of serum chemistries in rats.

Power densities: 0, 20, or 30 mW/cm^2.
Length of exposure: 120 min.
Exposure array: two cages.
Biologic assay: serum chemistry values immediately after exposure.

TABLE 2. COLONIC TEMPERATURES OF MALE CD RATS EXPOSED TO 2450-MHz MICROWAVES. EXPERIMENT 1

Exposure Duration (min)	Power density (mW/cm^2)		
	0	20	30
15	38.3 ± 0.2 (8)*	38.5 ± 0.3 (8)	38.6 ± 0.3 (16)
30	37.4 ± 0.5 (10)	38.1 ± 0.4 (10)	38.7 ± 0.6 (20)
60	36.9 ± 0.3 (8)	37.8 ± 0.5 (8)	38.9 ± 0.6 (16)
120	37.0 ± 0.4 (6)	37.3 ± 0.4 (6)	38.8 ± 0.5 (16)
180	37.0 ± 0.4 (6)	37.3 ± 0.5 (6)	39.0 ± 0.5 (16)
240	37.0 ± 0.3 (8)	37.3 ± 0.3 (8)	38.9 ± 0.5 (16)

*Values are means ± SD (N). Parenthetic numbers are population size.

TABLE 3. BODY WEIGHT AND COLONIC TEMPERATURES OF MALE CD RATS ACCLIMATED TO THEIR EXPOSURE CONDITION (0 mW/cm^2) FOR 90 min FOLLOWED BY VARYING EXPOSURE TO MICROWAVE RADIATION (30 mW/cm^2; 2450-MHz). EXPERIMENT 2

	Exposure duration (min) (N)			
	15 (16)	30 (16)	60 (16)	90 (8)
Before (°C)	38.0 ± 0.5*	37.7 ± 0.5	38.0 ± 0.4	38.2 ± 0.4
After (°C)	38.0 ± 0.4	38.1 ± 0.3	38.6 ± 0.6	38.7 ± 0.7**
Δ °C	-0.01 ± 0.5	0.4 ± 0.5	0.7 ± 0.8	0.5 ± 0.9
Body weight	412 ± 17	413 ± 23	416 ± 19	416 ± 15

*Values are means ± SD.

**p = 0.0002; df = 3,52; F = 8.03.

TABLE 4. COLONIC TEMPERATURES OF MALE CD RATS GIVEN ANESTHETICS FOLLOWED BY EXPOSURE TO 2450-MHz MICROWAVES, 30 mW/cm^2, FOR 90 min. EXPERIMENT 3

Drug (mg/kg)	Mean	SD	N
Anileridine, 10 s.c.	40.4	0.5	6
Chloral hydrate, 300 i.p.	39.0	0.9	7
Pentobarbital, 50 i.p.	39.9	0.5	7
Promazine, 40 i.m.	40.0	0.5	6

TABLE 5. RELATIVE AND MEAN VALUES (- TO +++) OF PHYSIOLOGICAL PARAMETERS IN CHEMICALLY-RESTRAINED MALE CD RATS: COLONIC TEMPERATURE CHANGE DURING EFFECTIVE PERIOD WITH AND WITHOUT IRRADIATION BY 2450-MHz MICROWAVES, 30 mW/cm^2 FOR 90 min. EXPERIMENT 3

Drug and dose	Depth of sleep and duration (h)	Lack of response to touch	Δ T (°C)	
			Without radiation	With radiation
Anileridine (10 mg/kg s.c.)	++ 1.5*	++	0	+3.0
Chloral hydrate (300 mg/kg i.p.)	+++ 2	++	-1.5	+3.0
Ketamine (160 mg/kg i.m.)	+ 1	++	-1.5	--
Pentobarbital (35 mg/kg i.p.)	+++ 2	+++	-2.0	+3.5
Promazine (40 mg/kg i.m.)	+ 1	+	-1.0	--
Xylazine (0.75 mg/kg i.p.)	- 0	+	-2.0	--

*N = 8.

TABLE 6. SERUM CHEMISTRY (SMAC-24) VALUES OF MALE CD RATS MICROWAVE-IRRADIATED FOR 120 min AT 2450 MHz, THEN BLED IMMEDIATELY. EXPERIMENT 4

	0 mW/cm^2*		20 mW/cm^2**		30 mW/cm^2*	
	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
Blood urea nitrogen (mg/dl)	14.1	3.2	17.6	3.3	16.1	1.7
Sodium (mEq/L)	141	10	147	1.1	147	1.8
Potassium (mEq/L)	5.97	1.0	5.76	0.42	6.15	0.97
Chloride (mEq/L)	94.1	5.5	98.6	1.5	98.6	1.7
Calcium (mg/dl)	11.0	1.3	11.3	0.52	11.0	0.61
Phosphorus (mg/dl)	8.00	0.66	7.81	0.32	7.31	0.58
Uric acid (mg/dl)	2.68	0.68	3.43	0.84	3.13	0.97
Cholesterol (mg/dl)	68.0	17	66.9	12	71.2	11
Triglyceride (mg/dl)	52.4	17	68.9	40	34.8	17
Alkaline phosphatase (mU/ml)	250	88	315	68	192	43†
SGOT†† (mU/ml)	67.8	16	76.0	16	76	14
SGPT†† (mU/ml)	31.8	7.1	38.3	8.1	37.5	7.9
CPK†† (mU/ml)	48.1	20	58.4	44	42.9	18
LDH†† (mU/ml)	196	156	328	218	213	105
Total protein (g/dl)	5.69	0.73	6.13	0.22	6.38	0.40
Albumin (g/dl)	3.17	0.38	3.40	0.16	3.44	0.19
Globulin (g/dl)	2.52	0.40	2.73	0.19	2.94	0.29
Albumin/globulin	12.6	0.13	1.26	0.09	1.19	0.14
BUN/creatinine	37.6	8.6	34.0	4.9	31.1	3.8
T_4 (µg/dl)	4.97	0.86	4.79	0.81	4.47	0.87
T_3 (ng/dl)	56.0	20.2	52.3	31	3.57	15
Corticosterone (ng/ml)	116	93	156	83	260	85†

*N = 10, except corticosterone and alanine transaminase (SGPT) = 9.

**N = 7.

†$p < .01$.

††SGOT = aspartate transaminase. SGPT = alamine transaminase. CPK = creatine phosphokinase. LDH = lactic dehydrogenase. BUN = blood urea nitrogen.

TABLE 7. SERUM CREATININE VALUES
AND THE ACTUAL AND RELATIVE INCIDENCE (% OF COLUMN)
IN EACH POWER DENSITY; $X^2 = 27.15$, $p < 0.0001$. EXPERIMENT 4

Creatinine (mg/dl)	Power density (mW/cm^2) 0	20	30
0.3	2 (20)	0 (0)	0 (0)
0.4	8 (80)	0 (0)	0 (0)
0.5	0 (0)	6 (86)	8 (80)
0.6	0 (0)	1 (14)	2 (20)

TABLE 8. SERUM TOTAL BILIRUBIN AND THE ACTUAL AND RELATIVE INCIDENCE (% OF COLUMN) IN EACH POWER DENSITY

Total bilirubin (mg/dl)	Power density (mW/cm^2) 0	20	30
0	4 (40)	2 (29)	2 (20)
0.1	6 (60)	5 (71)	8 (80)

TABLE 9. SERUM DIRECT BILIRUBIN AND THE ACTUAL AND RELATIVE INCIDENCE (% OF COLUMN) IN EACH POWER DENSITY

Direct bilirubin (mg/dl)	Power density (mW/cm^2) 0	20	30
0	10 (100)	6 (86)	10 (100)
0.1	0 (0)	1 (14)	0 (0)

TABLE 10. SERUM INDIRECT BILIRUBIN AND THE ACTUAL AND RELATIVE INCIDENCE (% OF COLUMN) IN EACH POWER DENSITY

Indirect bilirubin (mg/dl)	Power density (mW/cm^2) 0	20	30
0	4 (40)	3 (43)	2 (20)
0.1	6 (60)	4 (57)	8 (80)

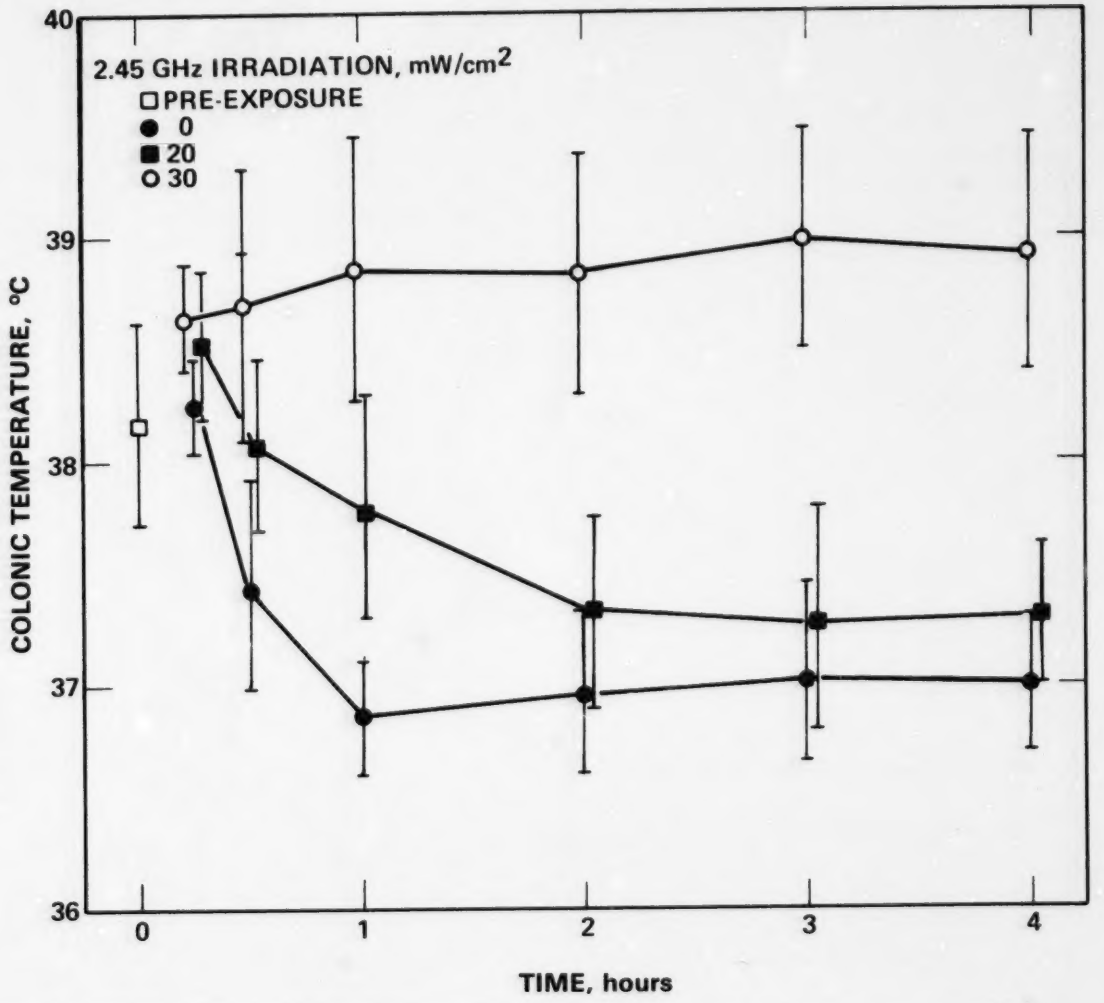


Figure 1. Colonic temperature in male CD rats following exposure to microwave radiation, Experiment 1.

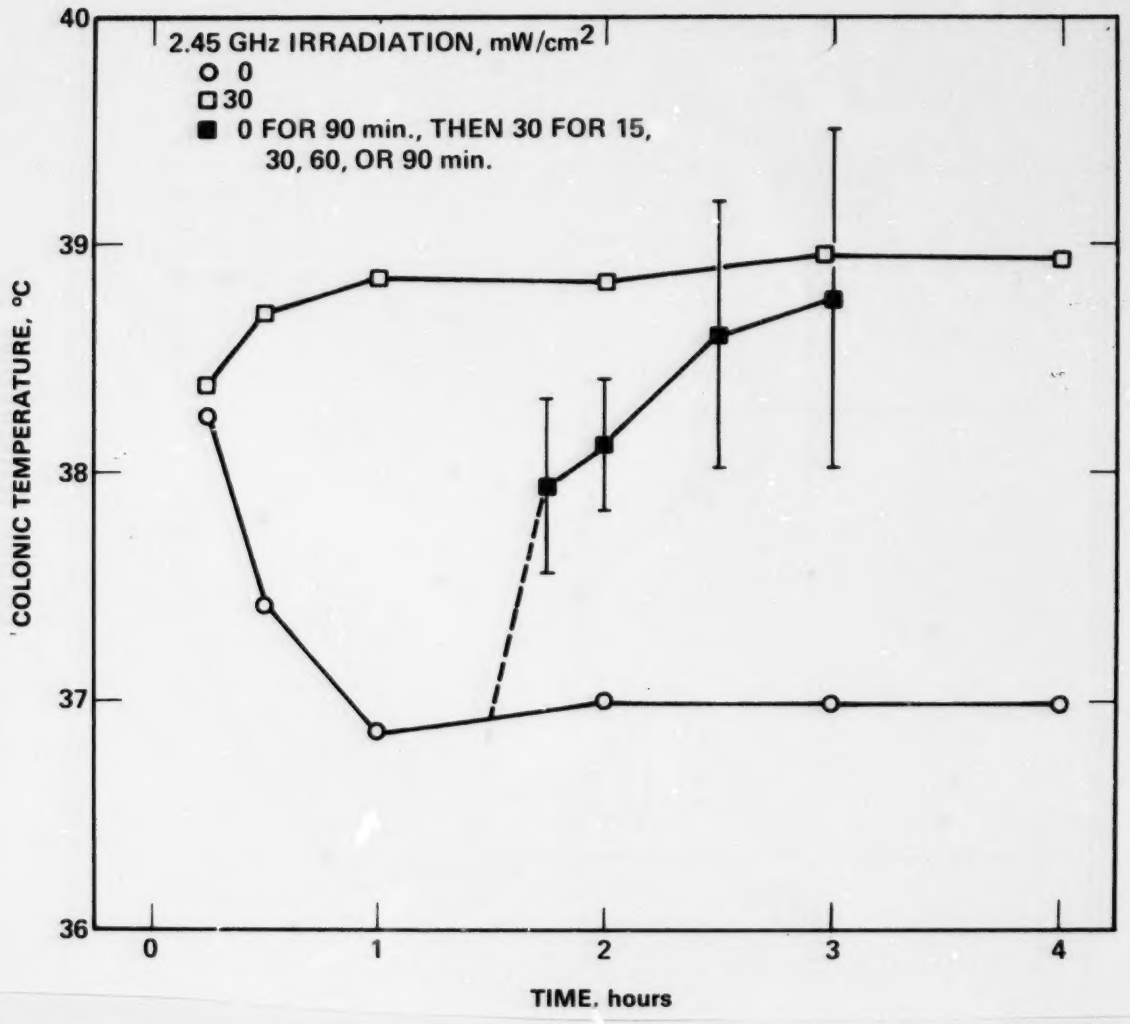


Figure 2. Colonic temperature in male CD rats following exposure to microwave radiation, Experiment 2.

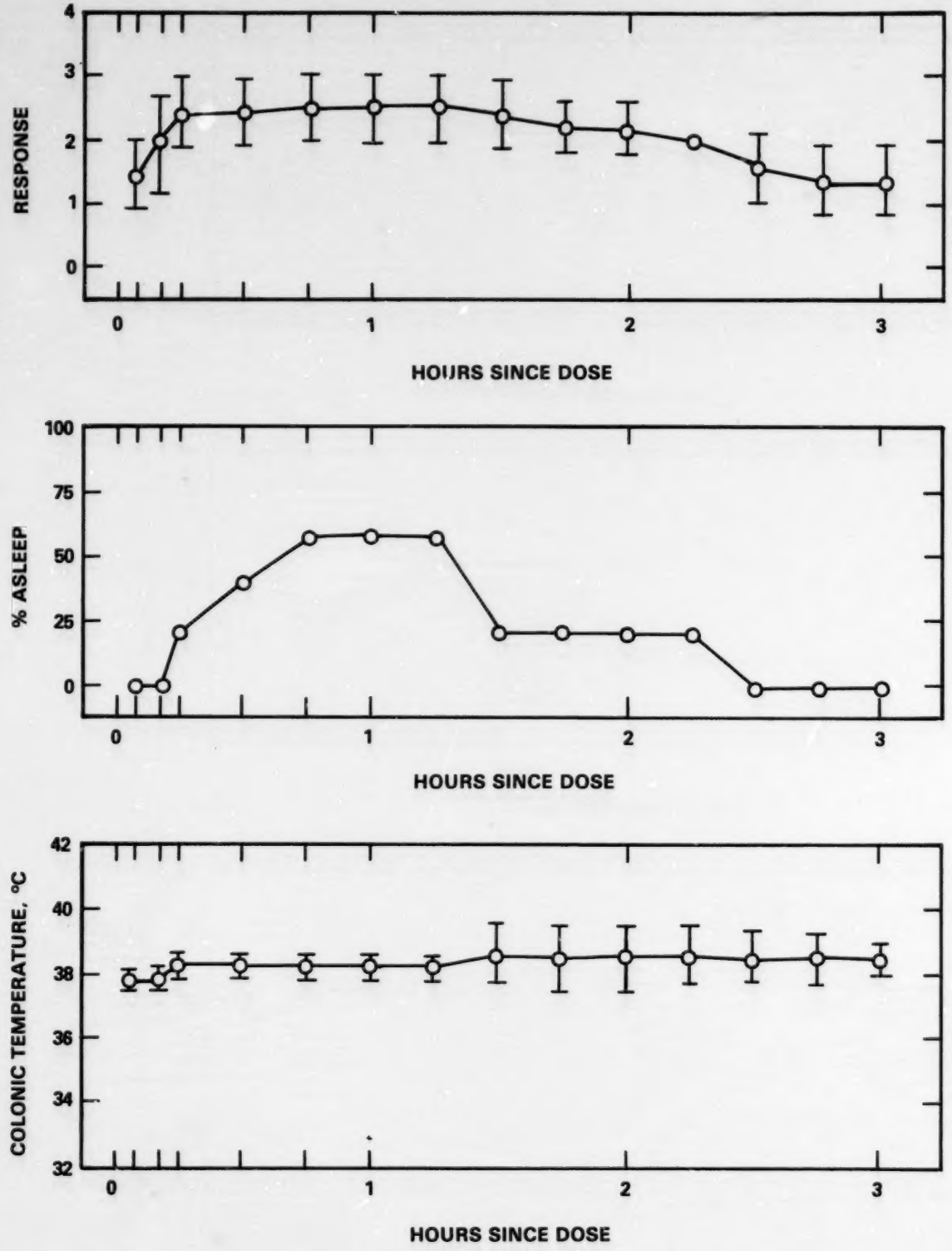


Figure 3. Colonic temperature, percent asleep, and responsiveness in male CD rats treated with anileridine, 10 mg/kg s.c., Experiment 3.

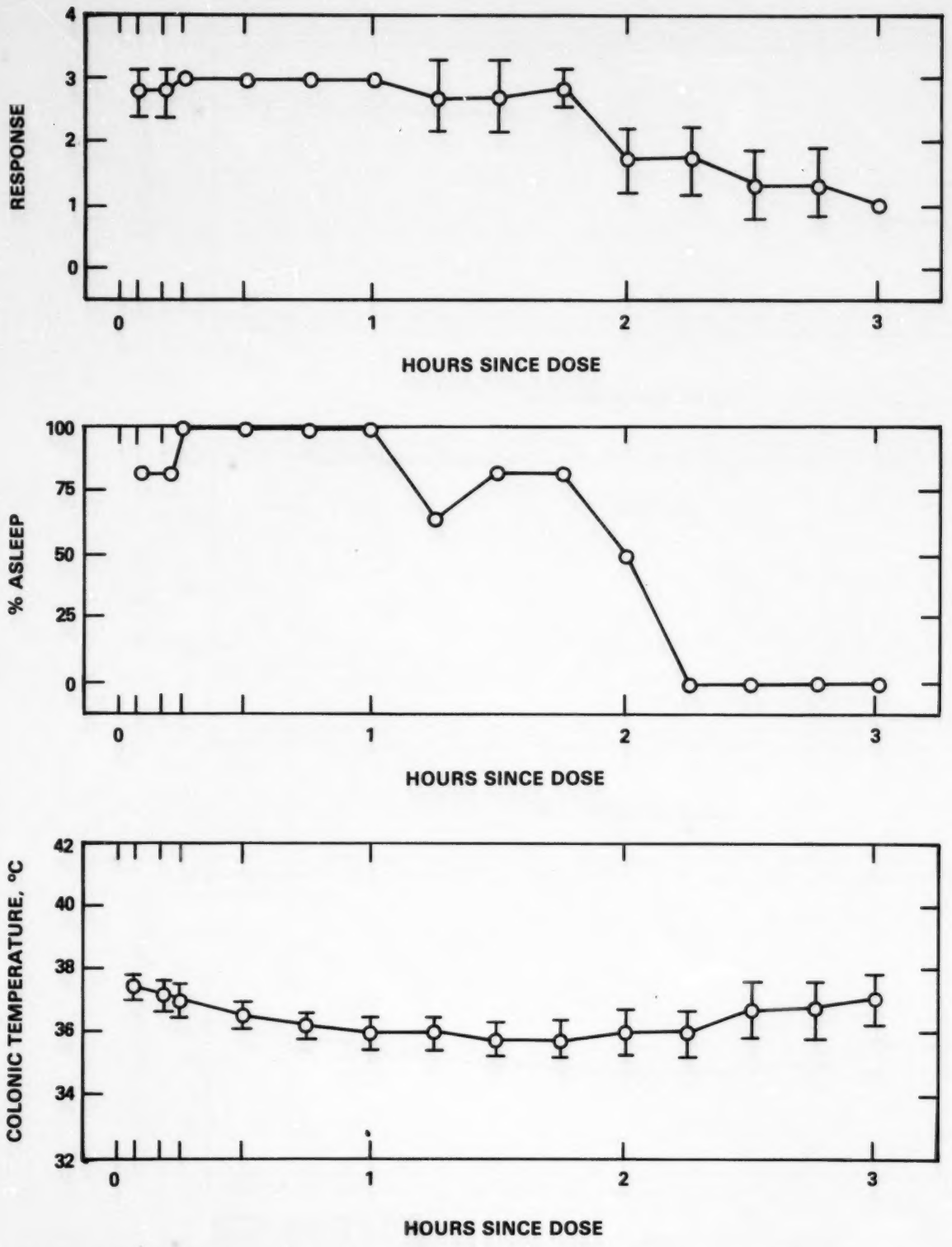


Figure 4. Colonic temperature, percent asleep, and responsiveness in male CD rats treated with chloral hydrate, 300 mg/kg i.p., Experiment 3.

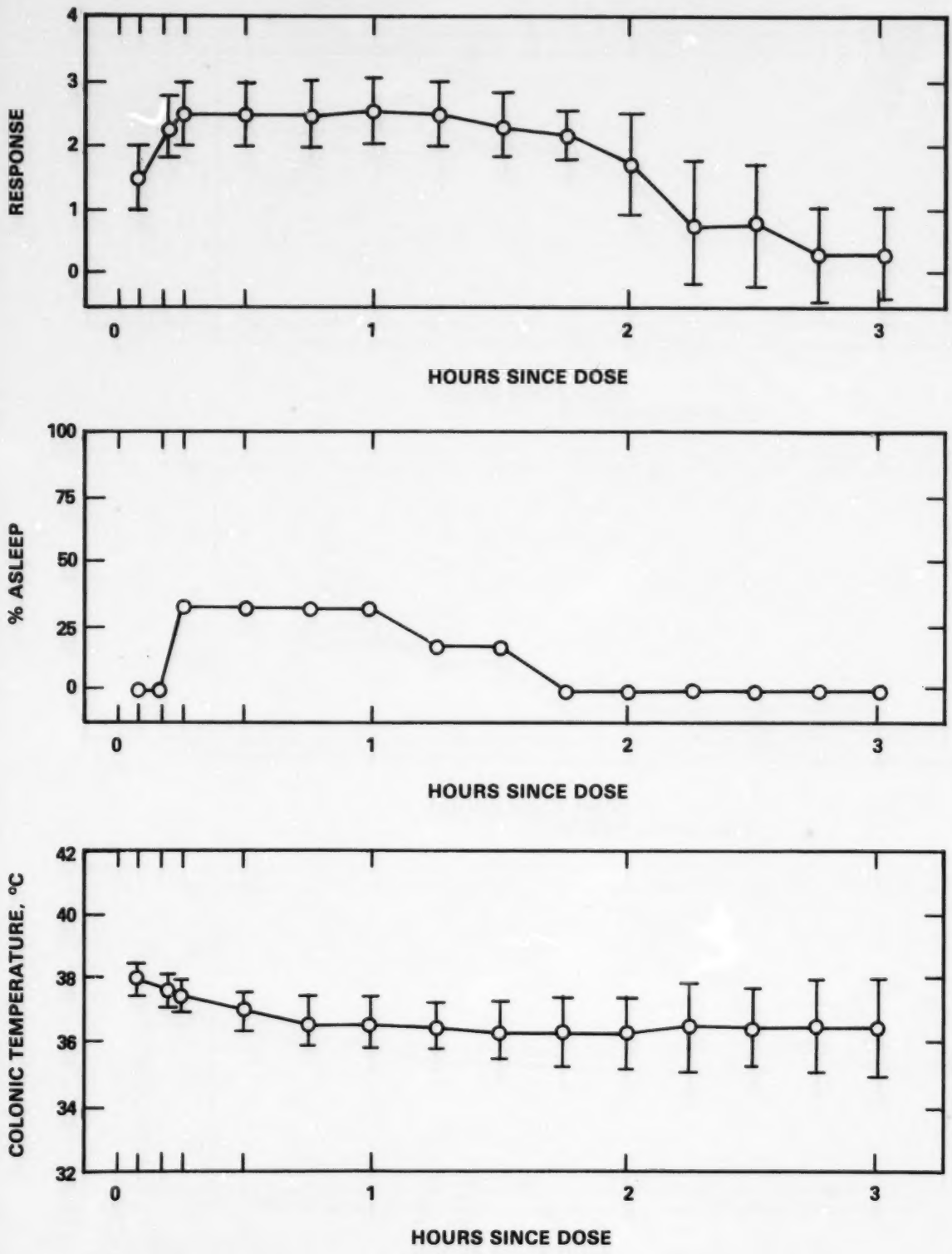


Figure 5. Colonic temperature, percent asleep, and responsiveness in male CD rats treated with ketamine, 160 mg/kg i.m., Experiment 3.

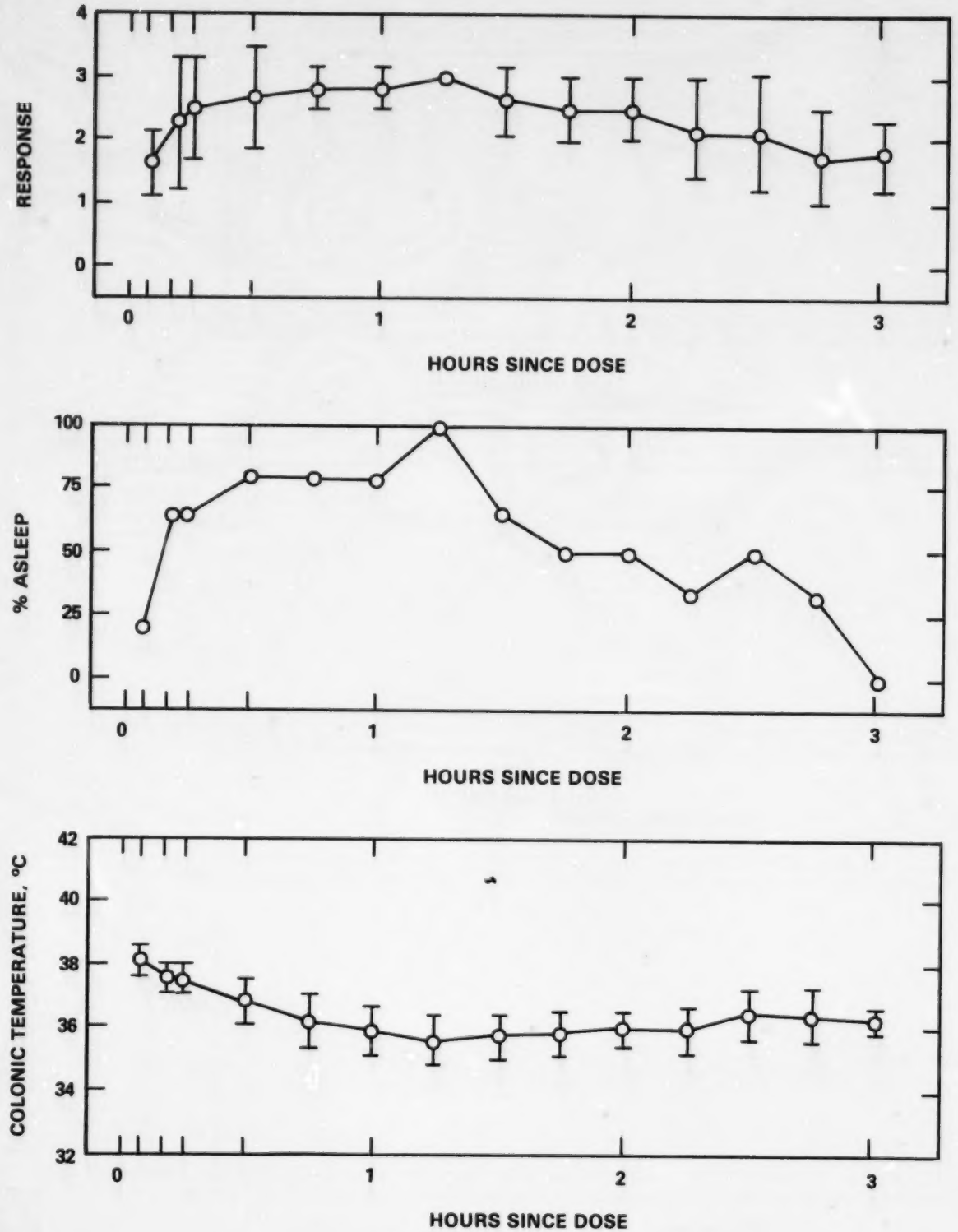


Figure 6. Colonic temperature, percent asleep, and responsiveness in male CD rats treated with pentobarbital, 35 mg/kg i.p., Experiment 3.

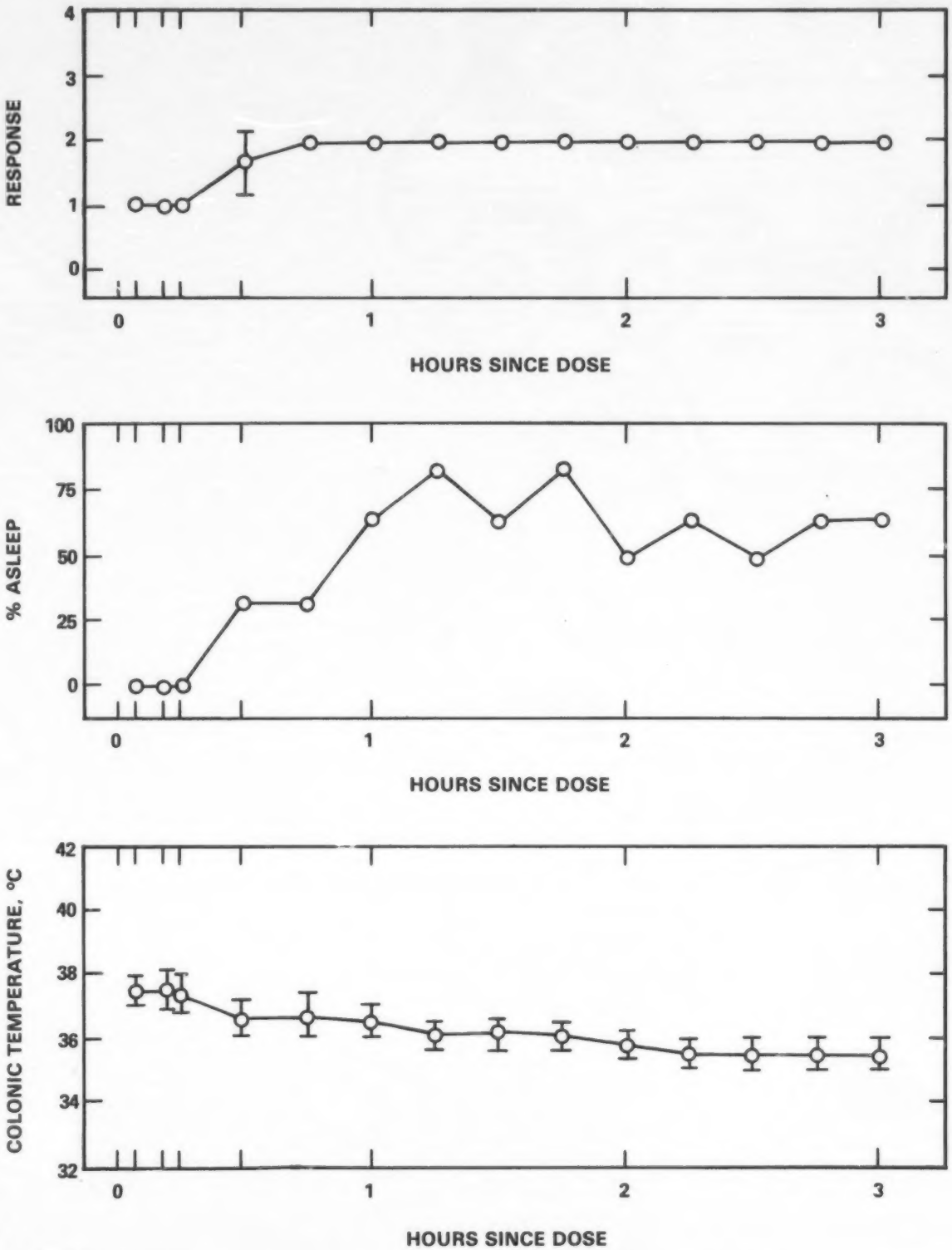
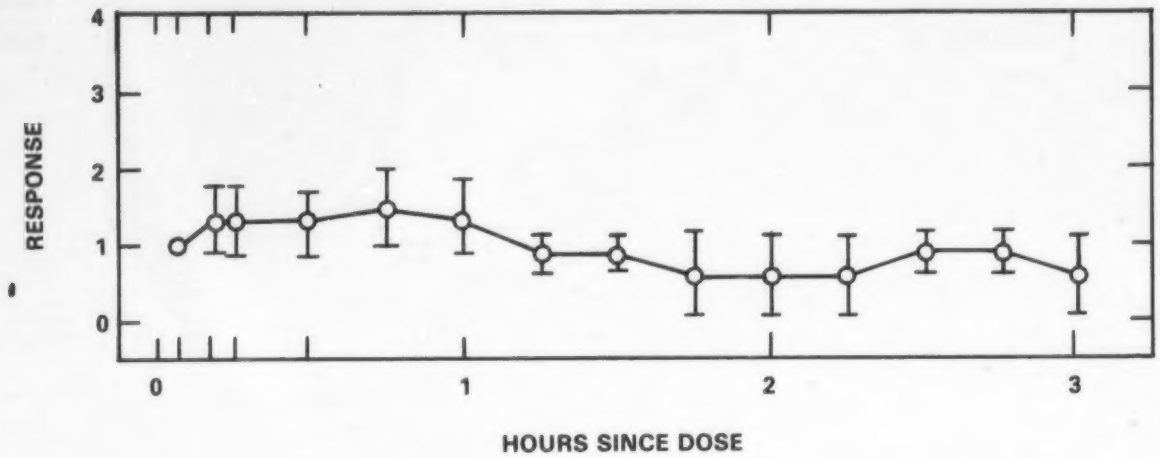


Figure 7. Colonic temperature, percent asleep, and responsiveness in male CD rats treated with promazine, 40 mg/kg i.m., Experiment 3.



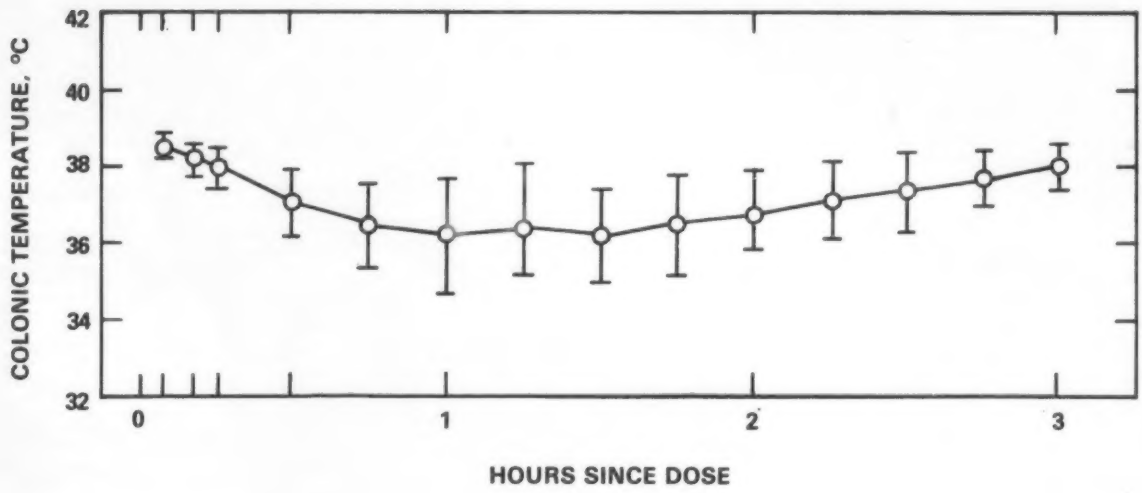


Figure 8. Colonic temperature, percent asleep, and responsiveness in male CD rats treated with xylazine, 0.75 mg/kg i.p., Experiment 3.

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